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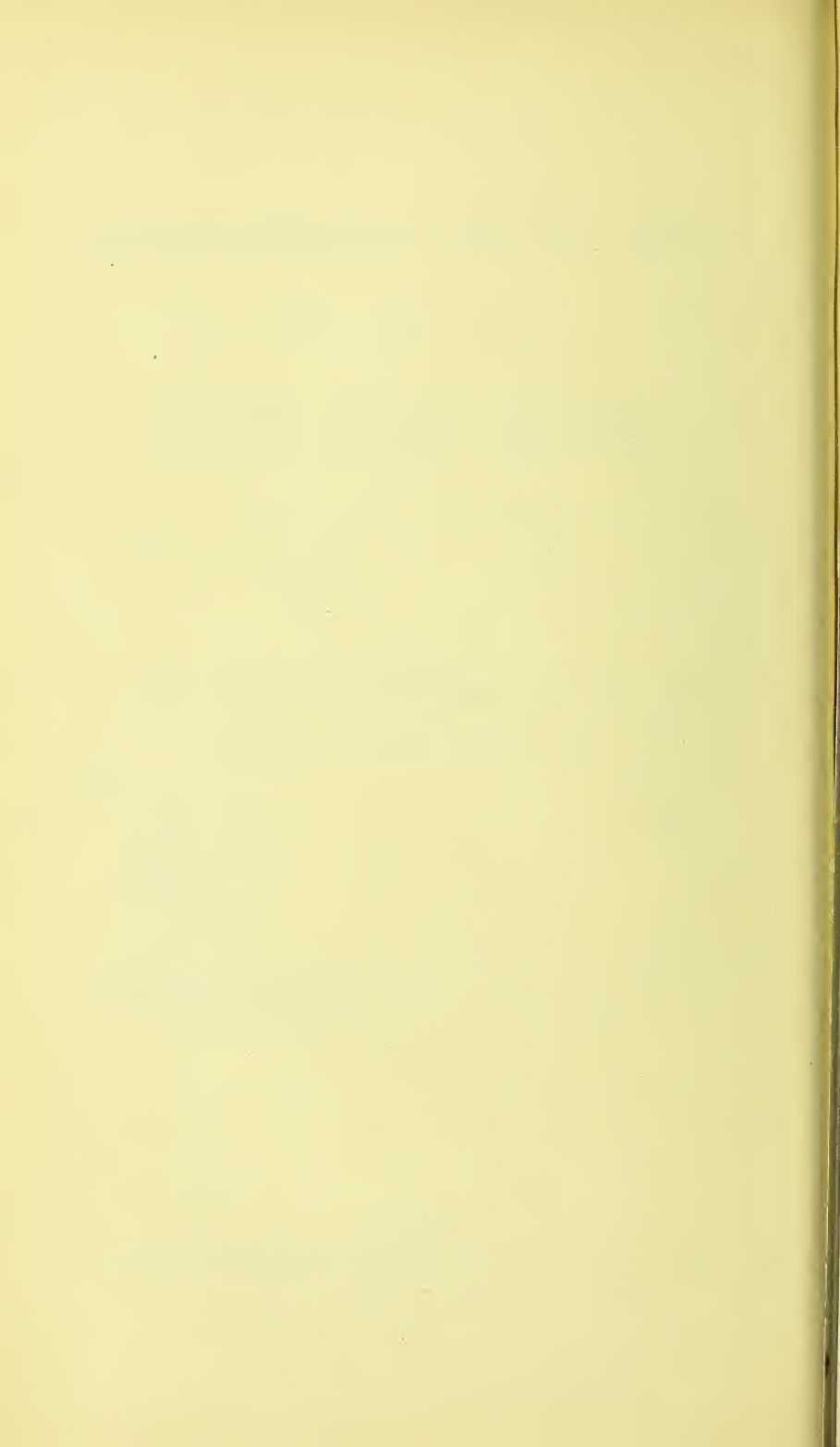
SOME RECENT MANIFESTATIONS
OF
CEREBRO-SPINAL FEVER.

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SOME RECENT MANIFESTATIONS OF CEREBRO-SPINAL FEVER.

BY A. K. CHALMERS, M.D., MEDICAL OFFICER OF HEALTH OF GLASGOW.

(Read: Friday, May 3rd, 1907.)

IN recent years the subject of cerebro-spinal fever has already occupied the attention of this Society on two occasions.

In 1899¹ Dr. Bruce Low reviewed the history of the prevalence of the disease, and last year (1906) Dr. Farrar² discussed it, with special reference to its infectivity.

Dr. Low believed there was reason for thinking that the number of deaths annually attributed to the disease did not fairly represent the numbers actually occurring; and he presented a chart which brings, I think, into striking contrast a decrease in the number of deaths attributed to cerebro-spinal fever in the decade 1881-90, with an increase in the number of those attributed to simple inflammation of the brain and membranes in the same period.

This contrast seems to me the more significant because, I have been watching during recent months a change in the registered causes of deaths in precisely the opposite direction, *i.e.*, a shrinkage in the number of deaths attributed to simple meningitis (if there is such a pathological entity) coinciding with an increase in the number of those correctly, I believe, termed cerebro-spinal fever.

There is appended to Dr. Low's Paper a suggestive note by Dr. Still and Dr. Washbourn regarding the pathological relationship between basilar meningitis in children and true cerebro-spinal fever; and it would seem a not-unlikely result of the increased attention which all forms of meningeal disease are now demanding, that we shall be able, in time, to revise our nomenclature and re-classify the several forms, not only by their symptoms, but also by the organisms with which they are associated.

The question is not wholly one of scientific precision, but of every-day utility. Is cerebro-spinal fever never present except in epidemic form? Is every outbreak a re-invasion, or only the result of exalted activity and

¹ *Transactions of the Epidemiological Society*, vol. xviii, p. 53.

² *Ibid.*, vol. xxv, p. 245.

virulence in an organism to which for a time we have become tolerant until something occurs to disturb the balance?

Whether it is always present is a question which continued notification alone can answer; whether the disturbance is one of external conditions, or an acquired susceptibility in individuals, is at the moment one of surmise. One recalls the recurring waves of influenza and the gradually rising death-rate from pneumonia, as at least suggesting probable influences acting in this direction.

Apart from such generalisations there is unhappily ample evidence that the disease is at present widely spread; and the extent to which it is present in this country is indicated by the fact that 119 local authorities in England and Wales, and 163, representing 81 per cent. of the population of Scotland, have included it among the notifiable diseases.¹ In Ireland it is now notifiable in 6 county boroughs, and 91 Urban and Rural districts. Twelve months ago it was not notifiable anywhere, although several isolated cases, and one or two limited groups, had been observed both in England and Scotland.²

OUTBREAK IN GLASGOW.

The presence of the disease was recognised in Glasgow in the spring of 1906, and it is with its subsequent development that I wish chiefly to deal.

I must here claim your indulgence if I present a very imperfect picture of this. For one reason the outbreak is still in currency, and any opinion expressed is subject to revision later on. Further, I have had to prepare these notes under very considerable pressure of other work; and, indeed, my chief object is to obtain your opinion on such apparent facts as I can present.

And the better to define the subject we are to discuss, I shall quote to you a description of the disease by Dr. Grimshaw,³ which fairly enough corresponds with what we are now witnessing, although there are some important differences which are of interest.

¹ For this information I am indebted to Mr. Power and Dr. Leslie Mackenzie, of the Local Government Boards of England and Scotland respectively, and to Surgeon-Colonel Flinn, Medical Inspector of the Local Government Board of Ireland.

² During the years 1874-82, no deaths were registered in Scotland as due to cerebro-spinal fever, but between 1883-1904 the cause of death was so named in 102 cases. Of these, 40 per cent. were in children under five, a much smaller proportion than in our present experience.

³ Quain's *Dictionary of Medicine*, 1st edition, vol. 1, p. 226.

Dr. Grimshaw's description, drawn, I think, from the disease as he saw it in Dublin, is as follows:—

“An acute epidemic febrile disease, characterised by sudden invasion, with extreme nervous shock, vomiting, excessive pain referred to the back of the neck and spine, spasmodic contraction of the muscles, excessive sensibility of the spine, and frequently delirium, accompanied by *purpuric eruptions, either circumscribed, raised hard, and shotty to the feel; or extensive purpuric spots or patches,*¹ *frequently accompanied by vesicular eruptions, usually of herpetic, but sometimes of a pemphigoid, character, and frequently purulent inflammation of the eyes.*”

“*Age.*—The disease usually attacks those approaching the age of puberty or in early adult life. It is not infrequent in young children.”

“*Occupation.*—It appears specially to attack recruits in the Army and Royal Irish Constabulary.² Excessive fatigue seems to predispose to the disease.”

It may here be remarked that in our present experience the rash is never hard or shotty, or, indeed, palpable to touch, and never larger than a split bean; and that fully half the number attacked are children under 10 years. Further, that while the early stages are characterised by extreme restlessness, the muscular state, later, is one rather of tonic rigidity than of intermittent spasm.

But, as bearing on the question of age incidence, I may here note that along with unmistakable evidence of increasing virulence which marked the second period of the outbreak in Glasgow, there occurred also an increase in the proportion of cases at older ages.

I have already indicated that what may be termed a pre-epidemic period occurred, and it will here be useful to indicate the clinical features of both periods. The following description was prepared for me by Dr. Dow, Senior Resident Physician in Belvidere, in September:—

DESCRIPTION OF CLINICAL FEATURES IN PRE-EPIDEMIC PERIOD.

“The patients are mostly children or young adults. The onset of the disease has been sudden, in all cases being

¹ This may correspond with the hæmorrhagic form of the disease which is described in other recent outbreaks, but we have had nothing resembling it.

² For reasons to be afterwards stated, it would have been interesting to know the cubic space per head allowed at that time in the Royal Irish Constabulary dormitories, but Surgeon-Colonel Flinn, after inquiry, tells me that there is no record.

ushered in with shivering, severe occipital and frontal headache, and in many cases with pain in the cervical and lumbar spine.

"*Vomiting* has been an early and almost constant distressing symptom.

"*Retraction of the head*, accompanied by painful stiffness of the muscles of the neck, has been practically a constant feature of the disease. The head may be so far drawn back that the occiput lies between the shoulder blades. In one case in the second period this over-extension reached to the nates.

"*Rigidity of the muscles of the back* is common, and in a few cases the back has been so greatly arched as to present the condition of opisthotonos.

"*Kernig's Sign* has been constantly present—viz., when the leg is raised in an extended position it suddenly becomes rigidly flexed, the flexor tendons at back of knee standing out prominently. The knee-jerks in some cases have been exaggerated, in others difficult to elicit. Plantar reflexes have been, as a rule, exaggerated. Ankle clonus has never been present. Of sensory symptoms, the most constant has been severe occipital and frontal headache, which persists from the outset throughout the disease in all the cases.

"*Great sensitiveness along the spine* and marked hyperæsthesia of the whole body has been present in many of the cases. Delirium has been usually present at onset, and sometimes of a maniacal type. It may give way to coma, or subside during the course of the disease. The pupils, as a rule, have been equal, and have responded to light, but in a few cases they are unequal, or have varied from day to day. Squinting has occasionally been observed. Nothing definite in the way of paralysis has occurred.

"The *face* has, as a rule, been flushed, suggesting pneumonia. Herpes has been common at the angles of the mouth and on the lips, and extensive herpetic eruptions have been found on the face, neck, shoulders and arms.

"A *petechial rash* has been observed, but only in a very small percentage of the cases, and has usually appeared within the first few days. The petechiæ have been found distributed all over the surface of the body, and have varied in size from the diameter of a pin-head to that of a threepenny-piece. The outline of these spots has been irregular. In one case a subcuticular mottling accompanied the petechiæ, somewhat resembling that of typhus.

"The *temperature* has been irregular and variable, but

usually of a remittent type. The pulse has often been markedly slow in adults, ranging from 40 to 50 per minute. In children the pulse has usually been rapid, and of low tension. The lungs, as a rule, have been free from lesion. Albumen has only occasionally been found in the urine. The spleen has never been found to be enlarged.

"Purulent discharges from eyes and ears have commonly occurred during the course of the disease."

DESCRIPTION OF THE CLINICAL FEATURES AT LATER PERIOD.¹

Since the above description, the disease has assumed a more acute or fulminant character, many cases ending fatally within a few hours of the onset.

An acute case has these symptoms:—A prodromal period lasting for an hour or two to twenty-four hours (rarely longer), during which the patient feels out of sorts, eats little, has some pains in the legs (a common prodrome), or has a mild headache and is dull; a rigor may be present or absent, and may precede by a few hours the actual onset.

The attack commences with vomiting or retching, a symptom which is never absent; or it may be preceded by a sudden pain in the head. The vomiting may be prolonged and recurrent, and the headache so severe as to at once launch the patient into wild delirium.

Next follows the irritative stage, with delirium, restlessness, nervous spasms, rigidity, screaming, frequency of micturition and vomiting, and contracted pupils. This stage may be absent or prolonged, and is followed by a comatose state, in which the patient is quiet when not disturbed; breathing becomes rapid and stertorous, face flushed and cyanotic. Death occurs in 24 to 48 hours, or longer.

Of the particular signs, stiffness of the head and Kernig's sign are the chief. The knee-jerks are active in most cases, and there is no ankle clonus. The pupils are contracted and fixed at first; later they are dilated, and fixed or sluggish. Squint is common.² Conjunctivitis is the rule, and the membrane is frequently hæmorrhagic. There is commonly a general spastic condition, but convulsions are

¹ Prepared by Drs. Brownlie and McGregor, Belvidere Hospital.

² Dr. McGregor adds that the squint is mostly spasmodic and variable, as opposed to being paralytic and fixed: a circumstance which plays an important part clinically in enabling the disease to be distinguished from tubercular meningitis. It is rare also to find optic neuritis.

rare; in some cases Jacksonian epilepsy has been present. A petechial rash¹ has been frequent, coming out a few hours from onset as a rule. It is distributed mainly on the abdomen and legs, although occasionally it is present on the face, and consists of subcuticular ragged hæmorrhages. Herpes has been more frequently present than the rash. The other features are much as formerly described.

TOTAL VOLUME OF MENINGEAL DISEASES.

With the introduction or recognition of a new cause of disease it is to be expected that some displacement will occur among those which present like clinical features. And if transference results, the precise effect of the new factor can best be appreciated by considering it in relation to the total volume of deaths in the class to which it belongs. In order to illustrate this, I introduce a Table of deaths from each of the three classes of meningeal deaths, yearly numbers being given for the years 1901-6, and those for the first quarter of 1907.

Glasgow.—Meningeal Disease: Number of Deaths Registered, 1901-6, and for first quarter, 1907.

	1901.	1902.	1903.	1904.	1905.	1906.	1907 (1st Quarter).
1. Cerebro - spinal Fever	1	147	312
2. Meningitis (simple), inflammation of the brain	438	414	411	388	363	329	39
3. Tubercular meningitis	237	244	240	260	237	307	105
	676	658	651	648	600	783	456

In the present instance, two most notable changes have taken place since cerebro-spinal fever was recognised. In the first place, the total volume of meningeal disease has increased, and the deaths attributed to simple meningitis have decreased. If we multiply the deaths in the first quarter of 1907 into an annual figure, those ascribed to

¹ The proportion of cases in which the rash was present varied at different periods of the outbreak, being present probably in the ratio of 1 to 5 cases during its worst period.

simple meningitis have decreased by one-half, and those attributed to tubercular meningitis have increased by a third, when compared with the figures for 1906.

This apparent increase in the number of deaths from tubercular meningitis is not supported by any other fact within my knowledge, and I think it suggests the inclusion of some which are due to cerebro-spinal fever.

But in what light are we to regard the shrinkage of simple meningitis? Has there been a transference to the cerebro-spinal fever group of many deaths which, without this disturbing factor, would still have found place under their original designation?

Meantime, until a more complete clinical picture of the symptoms which accompany each of the several forms of meningitis is forthcoming, bacterial verification alone can afford an answer, and this is not at all times possible. I have endeavoured to find one in a comparison of the morbidity rate of cases in hospital, verified and not verified bacterially, but which present similar clinical symptoms, with those similarly grouped at home, and the result for the 1906 cases may thus be stated:

Cerebro-Spinal Fever.—Cases and Deaths in Verified and Non-verified Groups.

1906.			Bacterially Verified.	Not Verified Bacterially. ¹
In fever hospitals	65	28
At home or elsewhere	22	77
			87	105
Deaths : ²				
In fever hospitals	47	20
At home or elsewhere	18	56
			65	76

—			Bacterially Verified.	Not Bacterially Verified.
Hospital	72 per cent.	71 per cent.
Home	82 per cent.	73 per cent.

All forms of meningeal disease have, unhappily, a high morbidity rate; yet I think in the foregoing Table we

¹ These were on clinical grounds regarded as cerebro-spinal fever.

² *Death-Rates.*

appreciate the true significance of the "non-verified" case, and with it the validity of the argument for transferring some deaths from the class of simple meningitis to the epidemic variety. Of 105 cases belonging to the "non-verified" category, 76 died, which is equal to a fatality rate of 72 per cent.; and the proportion of fatal cases scarcely varies, whether we take it on the hospital or home cases separately. And on comparing both with the fatal cases among the bacterially verified, the fatality rate corresponds almost accurately with that of the hospital cases, but falls short of that obtaining among the "home" cases, where the fatality rate is 82 per cent.

If we accept this as indicating that cerebro-spinal fever was present before it was recognised, and that deaths due to it were still being registered as simple meningitis, we may, by following the number of deaths registered in the several months of the year, put our finger on the precise point of time when the changes which we have been indicating occurred. And for this purpose it will be sufficient to deal in detail only with the deaths occurring under five years.

*Deaths Registered as due to Meningitis, Inflammation of Brain, etc.
Ages, 0-5 Years.*

Months.	Total Deaths, 1901-5.	Average Number Monthly.	1906.	Departures from Averages, July to December.	1907.
January	117	23	31	} + 23	14
February	102	20	31		6
March	124	25	20		8
April	138	28	27		—
May	132	26	32		—
June	125	25	29		—
	738	147	170	+ 23	
July	133	27	5	- 22	—
August	152	30	7	- 23	—
September	117	23	18	- 5	—
October	114	23	14	- 9	—
November	98	20	8	- 12	—
December	106	21	9	- 12	—
	720	144	61	- 83	
Total	1458	291	231	- 60	

On running down the line of figures under 1906, two facts are apparent. A change, which is scarcely a natural

one, began in July. The shrinkage which then occurs has no parallel in former years. During 1901-05 the deaths occurring monthly were distributed in a fairly uniform manner throughout the year, being on an average 123 in the first six months, and 120 in the second. In 1906, on the other hand, 170 deaths are attributed to meningitis during January to June, and only 61 in July to December. The increase in the first half of the year is quite as striking as the shrinking in the latter half, but its significance differs.

Turning now to the tubercular form of meningitis, we may also note some changes.

*Deaths Registered as due to Tubercular Meningitis,
Ages 0-5 Years.*

Months.	Total Deaths, 1901-5.	Average Number Monthly.	1906.	Departure from Average.	1907.
January ...	66	13	15	—	29
February... ..	74	15	13	—	24
March	76	15	16	—	27
April	95	19	14	—	—
May	83	17	15	—	—
June	62	12	12	—	—
	456	91	85	- 6	—
July	79	16	21	+ 5	—
August	64	13	25	+11	—
September	60	12	21	+ 9	—
October	73	15	18	+ 3	—
November	56	11	21	+10	—
December	68	14	27	+13	—
	400	81	133	+51	—
Total ...	856	172	218	+ 45	—

If we take as our basis for comparison the average number of deaths occurring in each half of the years 1901-05, we find some tendency towards a decrease in the incidence of the disease during the months January to June, but an increase from July to December. This difference is shown in 85 deaths in all being recorded in the first half of 1906, as against 91 in the corresponding portion of 1901-05; while from July to December the deaths attributed to it in 1906 number 133, as against 81 in previous years.

This, I think, may be taken as indicating that early in the

year the whole volume of meningeal disease underwent an increase which was at once recognised. At first it was regarded as simple meningitis, but when attention was directed to the presence of cerebro-spinal fever, many deaths were transferred to this category, and some, I think erroneously, to tubercular disease.

It is not necessary to continue this comparison into the later ages. After 20 the deaths from meningeal disease are relatively few in number; and changes quite similar to, but more limited in extent than, those occurring under 5 were observed.

The impression, therefore, left on my mind is that we have here some evidence for regarding cerebro-spinal fever as probably a constant element among our causes of death, but one which has had its virulence increased by some cause which we are not presently acquainted with.

DISTRIBUTION IN GLASGOW.

During 1905 I had, through the courtesy of Dr. Wallace Anderson, of Dennistoun, repeated opportunity of seeing with him several isolated cases of meningitis of a type¹ which he considers to have been present since 1891. No suggestion of grouping, however, occurred; nor were there any in which the organism was recovered, until the following incidents arrested attention:—

1. During March, 1906, two suspected cases were reported in a family residing in Main Street, Gorbals. Both were children, who sickened on the 10th and 13th respectively. The first child died after 22 hours' illness, the second after nine weeks. It was only after the disease was recognised in the children referred to in the following paragraph, that an effort was made to establish the accuracy of the diagnosis here by bacterial inquiry.

2. Meanwhile, in April, two children, aged three and eight, of a family which had recently arrived in town from Shettleston, sickened within 24 hours of each other. In the elder child death followed in four days; in the younger, after 22 days. These were the first to be verified bacterially in the Laboratory of the Public Health Department.

Coincidentally with these, a male adult, who had arrived in Glasgow from Bonar Bridge on April 7th, sickened on the 15th, and died on the 19th in the Royal Infirmary. From this patient, also, the organism of Weichselbaum was recovered.

¹ Chiefly owing to the occurrence of paralytic sequelæ, such as hemiplegia, deaf-mutism, etc. (See Notes by Dr. Anderson at end hereof.)

Other cases were also intimated from the Royal and Western Infirmaries and elsewhere; and by the end of May we knew of 23 probable cases, 2 of which sickened in March, 9 in April, and 12 in May. Of these, 16 proved fatal. In 3 cases, however, although death occurred in hospital in Glasgow, the patients had been admitted from Port Glasgow, Partick, and Whifflet, so that a considerable part of the Clyde area was already involved in the outbreak. Moreover, in the preceding winter (1904) cases had been recognised in one of the Clyde watering-places, and, almost coincidently with our own, cases were reported from Lanarkshire.

In 6 the organism of Weichselbaum was recovered. One death from meningitis occurred in a soldier, but the organism present was the *Staphylococcus aureus*.

Till the end of March, 1907, the movement of the outbreak falls into two periods—not equal in length, but sharply divided by the numbers occurring, by their distribution, and by evidence of an increased virulence of attack.

The second period began in the week ending January 12th,¹ and although up till the end of 1906 only three wards in the city had an absolutely free record, quite two-thirds of the cases were from the east and north-eastern portions, which have an estimated population of 334,179, or about two-fifths of the whole. Yet in none even of these wards did the cases occur in consecutive weeks. In some, indeed, they were separated by intervals of weeks. But when the increasing prevalence was established in January, weekly recurrences became more frequent, and in this respect the outbreak assumed one at least of the features of a true zymotic disease.

The following Table presents this altered incidence. A considerable portion of the increase in the second period

Wards.	Popula- tion.	1906. (June to December.)		1907. January to March.	
		Cases.	Per cent.	Cases.	Per cent.
1—9	...	134	67.3	251	53.6
9a—26	...	65	32.7	217	46.4
Total ...		199	100.0	468	100.0

¹ The mean temperature of the week ending December 29th was low, and this was regarded as having some bearing on the increased prevalence which followed. But the meteorological associations can only be adequately considered when the outbreak is over.

was supplied from the districts in which formerly the disease had been least prevalent.

CHANGES ACCOMPANYING INCREASED PREVALENCE.

The first most noticeable feature which accompanied the increased prevalence of the disease was the increased severity of attack, which I have already referred to in describing its clinical features. There was also a more frequent occurrence of multiple cases in families—an increasing tendency to invade families living under better housing conditions, and a lessening in the proportion of cases in infants under one year.

There is a tendency, I think, to regard the disease as being less influenced by insanitary conditions than most others with which the sanitarian is called to deal. Inquiring with regard to this some time ago, I quote some of the notes made at the time.

In one ward, out of 25 invaded houses, 11 were definitely dirty, although not filthy in the sense of administrative action. In another of 50 invaded houses, 12 were definitely filthy. In a third, with about 60 invaded houses, in 6 only has there been any degree of dirtiness. And again, of 40 cases all were in relatively-clean houses.

The general conclusion was that the houses invaded in any district reached the average standard of cleanliness for that district.

From this method of classifying them, it was impossible to suggest that the disease was invariably selecting the dirtiest houses; but when one discarded the general term "dirtiness" for one of pressure on air space, the question took a different complexion, which may thus be indicated:

Proportion of Cases occurring in Houses of several sizes compared with Proportion of Population living in them.

—	(1.) 1906. Proportion of Cases Occurring.	(2.) Proportion of Population Living in Houses of Various Sizes. (Census, 1901.)	(3.) 1907. January to March. Proportion as in Column (1).
1-apartment	22	14	18
2 "	62	47	61
3 "	14	21	15
4 " and upwards	2	18	6
	100	100	100

It would be possible to establish a similar excessive incidence in houses of smaller size for almost all the infectious diseases, and for some which are not infectious; but the comparison, I think, suggests that of all the conditions which may be classed as insanitary, that of which the system is least tolerant is misuse of the air of respiration. Indeed, there occurred one instance, to which I shall again refer, where, in a densely-overcrowded house, the disease exploded with an intensity which recalled typhus fever, and attacked fatally 5 out of 10 inmates. Here there was a family of 10 persons, of whom 5 were adults, living in a two-apartment house, containing just over 2,100 cubic feet.

Support to the suggestion that air impurity or defective ventilation promotes the spread of the disease is afforded if we compare the number of inmates of invaded houses with the average for each size; for in the one-apartment houses which were invaded there were more than 4 persons (4.3) as against 3.183 for all houses of this class; in the two-apartment houses which were invaded the average occupancy was 6 persons as against less than 5 (4.927) for all houses of this class; and in the three-apartment houses which were invaded the occupants averaged 6.7 against 5.409 in all houses of three apartments.

*Number of Inmates in Invaded Houses compared with Average
Number in all Houses of corresponding size.*

Size of Houses.	Invaded Houses, 1906.	Average Occupancy. (Census, 1901.)	Invaded Houses, 1907. (January to March.)
1-apartment ...	4.3	3.183	4.1
2 ,, ...	6.0	4.927	6.1
3 ,, ...	6.7	5.409	7.4

Age-Distribution.—The age-distribution of the disease is of some importance. Dr. Grimshaw's statement is that it is common in those approaching puberty or in early adult life, although not infrequent in children. It would appear to have been mainly a disease of early adult life in several outbreaks; and from Dr. Porter's Report (1906) on the Health of Johannesburg, I find that in the recent outbreak there it attacked mainly white children and Kaffir adults, the latter being employed, I think, in certain of the mines.

There are obvious objections to comparing different out-

breaks on the basis of attacks only, but in the following Table I have adopted this method of stating the proportions given by Dr. Taves, of New York,¹ in his analysis of the Gouverneur Hospital cases, and comparing them with our own.

Percentage Age-Distribution of Cases.

—	-1.	-5.	-10	-15	-20	-25	-35	-45	-55	55 +
Fiske Fund Essay ...	9	38	23	21	6	3	oldest	25 years		—
Glasgow, 1906 ² ...	18	38	20.5	10.2	4.4	3.9	2.9	.9	.9	—
Glasgow, 1907 ² (January to March)	14.5	38.1	18.2	11.6	5.5	2.8	5.5	2.8	1.7	—

Comparing both with the proportion (71 per cent.) given by Hirsch of 1267 fatal cases, New York has 91 per cent., and Glasgow 87 and 82 per cent. of cases, at ages 0-15.

The proportion of cases among infants under one year has some bearing on methods of infectivity; and here we may only note that in Glasgow we had 18 and 15 per cent. against 9 in New York.

Nature of Food in Infants under One Year with Relation to Method of Infection.—At an early period the condition of the intestine in fatal cases arrested attention by the presence of patches of congestion in the intestinal canal. These were situated sometimes in the duodenum, but most commonly towards the lower end of the ileum, where the congestion might be confined to Peyers patches, or be more uniformly distributed. Did food play any part in the spread?

One or two cases brought definitely into view the occurrence of quite temporary diarrhoeal disturbances, either in the patient or in others of his family, preceding the definite onset of the disease; and this led to systematic inquiry for minor disturbances in health in the inmates of invaded houses. But no mass of evidence in this direction could be found, and efforts to recover the meningococcus from the intestinal discharges or in the bowel wall have not hitherto been successful.

But on the general question I am disposed to regard the relative infrequency of simultaneous attacks in families, and

¹ Fiske Fund Prize Essay.

² Subject to revision, as all cases registered as cerebro-spinal fever are included in the figures.

the occurrence of the disease in children who, after inquiry, one can regard definitely as being wholly breast-fed, as setting aside food as a common vehicle of infection—if it is indeed one which is ever operative—and of this we have no evidence.

In one or two instances of breast-fed babies, however, the occasional soiling of “dummy teats” was noted as affording a possible method of mouth infection.

I may here place on record the age and character of the feeding in the case of 43 infants, where it was carefully investigated.

Cases Occurring among Infants under One Year, and Nature of Feeding.

Age in Months.			Total.	Nature of Food.
— 3	— 6	— 12		
2	10	12	24	On Breast alone. ¹
—	6	5	11	On Artificial Food alone—(Milk, condensed & sweet, Bread.)
—	2	6	8	On both (Porridge—Milk, condensed & sweet.)
2	18	23	43	Total.

Multiple Cases in Households.—I have already referred to one instance when the disease behaved in an overcrowded house much after the manner of typhus fever. There were others of a like—although less pronounced—form; and it rapidly attracted notice that in the second period of our outbreak these occurrences were more numerous than during 1906. It was regarded, indeed, as evidence that the organism was acquiring an increased virulence. And yet, the most striking feature of these incidents is that they are relatively infrequent. Of 186 households invaded during 1906, there were 6 only in which 2 cases occurred, and 1 family had 3 attacks. There is thus something less than 4 per cent. (3.8) of multiple attacks in this period; but during January to March, 7.5 per cent. of the invaded houses had multiple attacks, there being 26 households in which 2 cases each occurred, 3 households had 3 cases each, 1 had 4, and another 5.

It was difficult at first to find any factor which appeared to determine the greater incidence in these houses. The special susceptibility of children, as indicated by the

¹ One 20 days old; one 8 weeks old.

excessive proportion of cases at ages under 10 years, was of course obvious, and so our multiple cases usually occurred where children were numerous. But when the question of air impurity emerged, the actual number of inmates in invaded houses assumed greater importance; and it was found that an approach to grading the number of inmates in relation to the number of attacks therein could be made.

This may be indicated in the following Table.

Number of Persons Attacked per House.	Inmates per House.			
	One Apartment.	Two Apartments.	Three Apartments.	Four Apartments.
1 each	4.2	5.8	7.4	7.2
2 „	5.0	7.0	7.5	7.5
3 „	5.0	8.0	—	—
4 „	—	8.0	—	—
5 „	—	10.0	—	—

In some cases the difference in the number of inmates is small, but the figures seem to me to suggest that air impurity is related both to the question of invasion of particular households, and to the possibility of multiple attacks occurring.

In these multiple invasions, however (31 houses in all), it was found that a certain number occurred at varying intervals *after* the termination of the earlier case and disinfection of the house.

Of such there were 17; but leaving aside 3 third cases and 1 fourth case, there were 13 secondary attacks occurring after the following intervals.

Interval between Disinfection of House and Sickening of Subsequent Cases.

—	Days.							Weeks.			Month.	
	1.	2.	3.	4.	5.	6.	7.	2.	3.	4.	2.	3.
No. of Cases ...	2	1	1	1	1	—	—	3	—	—	4	—

We may accept the 6 cases sickening within 5 days as due to direct contact with the sick, and should probably include with them those who sickened in the second week. The possibility of re-invasion cannot, of course, be excluded; but in the case of 11 persons not residing in already invaded houses, there was known contact of limited duration, and in 5 of these the interval between exposure and sickening extended into the second week.

The following details of the circumstances under which this limited contact occurred, and the interval between it and sickening, were ascertained.

“Contact” Cases, excluding those among Inmates of already Invaded Houses.

Nature of Contact with Primary Case.	Interval in Days between Contact and Sickening.	Remarks.
1. Playfellow ...	6	Last contact on day of sickening of first patient.
2. Visitor ...	10 to 8	Primary case died on second day of illness.
3. Ditto ...	6	Visited primary patient on day of sickening.
4. Playfellow ...	2	Saw patient before sickening, and was possibly infected from same source.
5. Visitor ...	9 to 6	Visited on two occasions at interval of three days.
6. Ditto ...	A few hours	Visited patient in hospital on day of own sickening.
7. Ditto ...	11	At same school, and visited during illness.
8. Ditto ...	3	---
9. Ditto ...	2 to 1	Visited house at death of primary patient, and saw body.
10. Ditto ...	14 to 7	Visited several times during illness of primary patient, which lasted seven days.
11. Playfellow and visitor during illness	1	---

Some of these cases suggest that the period of incubation may be less than a day, but the occurrence of secondary cases in the second week, and more especially in the second month, after known exposure, raises broadly the query whether the infecting agent is present only in the persons of those attacked, and is not sometimes present in regions which disinfecting processes do not reach.

"Carrier" cases have been recognised in diphtheria, and in cerebro-spinal fever elsewhere they would also appear to have been demonstrated. We have succeeded only in a few instances in recovering the organism from the naso-pharynx, although the late occurrence of secondary cases suggests that some such method of transmission is much more frequently in operation.

Other factors may come into operation in determining the occurrence of multiple cases. We have seen reason for thinking that they may be related to conditions of air impurity. Dr. Farrar suggested, I think, the co-operation of the meningococcus with other organisms; and remembering the exceedingly active growth of round-cell tissue in the naso-pharynx in childhood, the special incidence of the disease in these years may be explained. But if the meningococcus acquires virulence by this co-operation, it would appear to take the form of an exalted activity, because mixed infections are rare. At Belvidere Hospital two only have been observed. In one case, Dr. Brownlie tells me the meningococcus was found in association with the tubercle bacillus, in another with a diplococcus. Moreover, in 106 examinations made by Dr. Buchanan, our bacteriologist (71 of which were by lumbar puncture after death, and 35 were autopsies), he found the meningococcus alone in 71 cases; the pneumococcus alone in 2 cases; both were present in 1 case, the tubercle bacillus in 4, and a streptococcus in 1; while in 27 the results were entirely negative.

School Associations.—In thirty-eight schools, single cases only occurred; in seventeen schools, there were 2 cases each; in six schools, 3 cases each; and in four schools, 4 cases each; *i.e.*, 27 in all had multiple cases.

In only five instances did it happen that two children in the same class were attacked. In one school both children sickened on the same day; in another, the interval extended to 11 days; in a third, it was 42 days, but midway in this interval a child in another standard also sickened. In a fourth school the interval was five days; but these cases formed the second and third of a group whose dates of sickening were respectively February 4th, March 7th and 12th (two cases). In the fifth school, the interval extended from January 7th to February 23rd.

Of the four schools in each of which four cases occurred, the dates of sickening in one have already been given. Of the three others, the dates of sickening in one were, January 24th (two cases, different classes); February 19th,

March 11th; in another, January 17th, February 5th and 26th, and March 3rd; while in the third school they were respectively January 7th, February 23rd, March 2nd and 16th.

If few of these dates suggest direct transmission of the disease from one child to another, it must be remembered that, so far as we know it clinically, the onset is very sudden, and usually prevents school attendance from the beginning.

Lateral Spread.—Were the disease possessed of a high degree of infectivity at comparatively short ranges, we should expect to find not only that multiple cases in families were numerous, but that the inmates of other houses in invaded tenements were also being attacked with a fair degree of frequency. This is scarcely our experience, however, for in 23 instances only was more than one house in a tenement invaded. The intervals between these invasions were as follows:—

Intervals between Invasions of Separate Houses in same Tenements.

Intervals.	Days.							Weeks.				Months.						Total.
	1.	2.	3.	4.	5.	6.	7.	2.	3.	4.	2.	3.	4.	5.	6.	7.		
No. of instances	...	1	...	2	...	1	...	3	...	4	5	2	2	...	1	1	23	

In addition to these, there was one instance where two houses were invaded almost simultaneously, *i.e.*, within 24 hours of each other.

Whatever view we may take of the cases occurring within an interval of not more than two weeks of each other, there would appear reasonable ground for assuming that those occurring at longer intervals than one month were due either to reinvasions, or to the propagation of the disease by cases not coming within our clinical knowledge of it, and not indeed clinically recognisable as such at the present time.

Occupation.—No special incidence occurred which need be noted. The adult cases occurred mostly among the artisan section of the population.

Fatality Rate.—In such cases of the disease as were recognised, the death-rate must be regarded as excessive. This, of itself, may suggest that modified cases occur which are not recognised; and in the classification here adopted, there is a difference amounting to 20 per cent. between the

death-rate of those where bacterial verification was possible, and of those who on clinical grounds were regarded as suffering from the disease, but where the diagnosis could not be verified in a similar manner.

I have limited the statement to those only who were treated in hospital, in order that greater accuracy in the comparison may be obtained.

Cerebro-Spinal Fever: Deaths and Death-Rates in Cases treated to a termination up to March 31st, 1907. Hospital Cases only.

—	Totals.	Verified Bacterially.	Not Bacterially Verified, but on Clinical Grounds regarded as Cerebro- Spinal Fever.
Cases	312	175	137
Deaths	230	145	85
D / R. per cent. ...	73.7	82.9	62.0

Summary.—Such, then, are some of the more outstanding features of the disease with which we are dealing, and a few words may serve to summarise them.

The outbreak consists mostly of single cases in separate households, distributed over a wide area. It occurs chiefly in children who are related to each other with regard to date of sickening, but have, as a rule, no common centre of infection which is discernible.

Under ordinary conditions, therefore, it would appear that cerebro-spinal fever is possessed of a limited range of infectivity; that it spreads, in the majority of instances, through the agency of an intermediary—like the “carrier” case in diphtheria—but that under conditions which tend to produce air impurity in confined spaces it can assume the characters of a true air-borne disease—like typhus fever or measles—and attack a considerable proportion of persons breathing the same air.

The much-debated relationship of insanitary conditions to the spread of the disease would also appear to resolve itself into a question of the condition of the air surrounding the patient: when the space is limited and the air impure, the power of direct infection is greatly intensified.¹

¹ What appears, indeed, to be a striking illustration of the infecting power of the disease, even among persons massed together in the open, is afforded by comparing the numbers sickening in the weeks ending April 27th (during which there was an important public gathering in Glasgow and district) and May 4th with those in the preceding and following weeks. For the several weeks ending April 6th, 13th, 20th, 27th, May 4th and 11th, the numbers sickening were respectively: 35, 37, 36, 54, 41 and 23.

Secondary cases occur, but *contact-groupings* are comparatively rare. On the other hand, on a spot map *place groupings* without any other associations are suggested.

When *multiple* cases occur, the sickenings may take place almost simultaneously, or with only a day or two between them; but they may, on the other hand, be separated by an interval of weeks.

None of the medical or nursing staffs of the hospitals have contracted it, but Dr. Archibald, one of my assistants, sickened on the sixth day after visiting a house in which the disease was present, and when the smell nauseated him.

It would therefore appear that the degree of infectivity is influenced both by the susceptibility of the person exposed and by the conditions of exposure.

It may be that exalted activity of the naso-pharyngeal tissue in young children helps to determine the special incidence on them, or that the more prevalent habit of kissing children affords the medium by which the organism is transferred in them from "carrier" cases. In any case, it is suggestive that the disease is less prevalent after the age at which the activity of the naso-pharyngeal tissue ceases.

If we leave the individual, and consider the conditions surrounding him—and presumably predisposing him to infection—that of air impurity, and particularly impurity from respiration, seem to play an important, if not the most important, part.

But as neither susceptible persons nor unsuitable surroundings will originate an attack, what we have to consider is by what means the disease will reach, not only unrelated members of a community but remote rural districts.

The disease recurred in some of our houses after we had disinfected them; and carefully as the details of disinfection were scrutinised, we could find no reasonable ground for thinking that anything had escaped the process, save the inmates, who were not obviously ill.

Although nasal discharges have been from time to time examined, the organism was recovered only three times (14 cases) from patients by swabs taken through the anterior nares. It was recovered, however, from the naso-pharynx of four contacts, and it is suggestive that these were inmates of the houses in which four and five cases respectively had occurred.

"Carrier" cases and spray-infection would completely

explain the method of spread, were the organism recoverable in a larger proportion of contacts.

At present, we know little of the disease clinically, except in its most malignant forms.

CLINICAL NOTES BY DR. J. WALLACE ANDERSON, DENNIS-
TOUN, GLASGOW, ON CEREBRO-SPINAL FEVER.

The cases which are noted hereafter have occurred at different intervals since 1891, and their importance at the moment would seem to be due to the fact that they offer illustration of children who were attacked by cerebro-spinal fever at a time when it was not generally recognised as being present in this country.

Symptoms.—General Description.

These may be stated as fever, restlessness, vomiting (not so persistent as in the tubercular form), suddenness of onset, then convulsive seizures and tonic spasms, much intolerance of being examined or handled, even when coma is beginning. *The most striking clinical symptom is tonic spasm of the nuchal muscles, with retraction of head, which cannot be replaced without great pain.* Later, when effusion becomes established, and there is coma and Kernig's contraction, the picture is very marked.

Illustrative Cases.

- Case 1.*—September, 1891, J. McC., male, aged 10 months. Symptoms here were at first believed to be due to tubercular meningitis, but were much prolonged and ended in recovery. Emaciation very great. Had well-marked retraction of the head, tense fontanelle, squinting, tonic and clonic spasms, prolonged coma. Was four months more or less under treatment, but recovered completely.
- Case 2.*—1893, J. L., female, 14 months. Again was at first believed to suffer from tubercular meningitis, until duration of illness rendered this unlikely. Was comatose for nearly three weeks. Complete recovery.
- Case 3.*—1896, M. Q., female, 7 years. Came home from school sick and vomiting, complained of great pain in the head, and was soon (same day) comatose. Had the symptoms already described, and was many weeks ill. Recovery very slow, but ultimately complete.

- Case 4.*—1897, C., male, 10 months. This is the first child with a record of permanent injury. He was 10 months old at time of attack; retraction of head very marked, with very tense fontanelle; coma, squinting, and Kernig's sign. Was many weeks between life and death, but got slowly better. Became deaf during attack, and has since been deaf-mute, though otherwise a bright and clever lad.
- Case 5.*—1898, K. M., female, 3 to 4 months. Was seized during evening; violent pain in head, required opiate to control screaming. Coma followed and continued for several days. Later had hemiplegia of right arm and leg, paresis of deglutition, inequality of pupils and feeble response to light; still later, choreic movements in the right arm developed, and which still continue, associated with much muscular wasting. Here the fontanelles were not tense, suggesting that the effusion had become sacculated. Made slow and imperfect recovery.
- Case 6.*—June, 1899, I. M., female. Was seized at Prestwich, Ayrshire, and in a day or two brought home to Glasgow. Symptoms vague at first, then the contractions became established. During height of illness, a deep-seated abscess in femoral region was opened under chloroform. Was long ill, but has completely recovered. Now eight years of age.
- Case 7.*—September to November, 1901, H. McK., 2 months, female. Had all the symptoms, convulsions, tonic spasms, coma, emaciation in marked degree. Seemed moribund and hopeless for weeks, and as if brain would never revert to normal; yet now at age of 7, there is no evidence of attack, except bad articulation.
- Case 8.*—June to August, 1902, A. C. B., female, 3 months. Had usual sudden onset, with well-marked symptoms, fulminant in type; early left pneumonia, with a marked consolidation. Very marked degree of muco-enteritis, with bloody stools; marked retraction of head and long-continued coma. Was very ill from June 6th to August 30th—eighty-five days—but made complete recovery.
- Case 9.*—J. D., male, 2 years. Symptoms began with general convulsions, necessitating the administration of chloroform. Ordinary contractions, with Kernig's sign, soon supervened. During prolonged coma he emaciated rapidly, and on recovery was discovered to be deaf.

He is now being educated at the Deaf and Dumb Institution, otherwise bright and healthy boy.

Case 10.—February, 1905, Baby Fraser, female, 7 months.

Had convulsions, followed by well-marked contraction in left arm and leg. Later on, hemiplegia on other side. At time of this child's illness four others were ill in the street where she resided (two of them, living in one lane, were cousins), and one in an adjacent street. Thus there was a suggestion of place-grouping.

Case 11.—December, 1905, A. C., male, 1 year. Was seen in consultation with Dr. Lindsay Steven, who agreed as to its character. Rectal temperature ran between 104 and 105. Kernig's sign present. After temperature became normal, child weak and very nervous, shrinking from the approach of anyone. Complete recovery.

Case 12.—July, 1906, S., male. Born in 1904. Symptoms not marked at first. Child emaciated progressively; seemed almost moribund for about two months, then came slowly round, and was then found to be totally deaf.

Case 13.—1907, C., female, 4 years. Became suddenly ill, with pain in head, vomiting, and coma. Retraction of head and high fever; coma passed off in from 4 to 5 days. She seemed to be progressing, when it was observed that she was deaf. There had been no pain in head, but she was very giddy, and could not stand or walk for weeks. The striking feature in this case is that the course of the illness was short, and ended almost abortively, and yet deafness resulted.
